IN THE SPECIFICATION:

On page 4, please replace the paragraph beginning on line 11 and ending with line 18 with the following rewritten paragraph:

> Figure 1 is a drawing showing a physical map of the NPHS1 locus at 19q13.1 and genomic ~~organisation~~ organization of the NPHS1 gene. Figure 1A, is a physical map of the 920 kb region between markers D19S208 and D19S224. Figure 1B, is a diagram of overlapping cosmid clones spanning the 150 kb critical region containing the NPHS1 gene. Location of polymorphic markers are indicated by arrows. Figure 1C, is a diagram showing the location of five genes, NPHS1, APLP1, A, B, C, characterised and searched for mutations in this study. Figure 1D, is a drawing showing a schematic structure of the NPHS1 gene;

On page 5, please replace the paragraph beginning on line 12 and ending on page 6, line 3, with the following rewritten paragraph:

> Figure 4 is a diagram of the Nucleotide-derived amino acid sequence of nephrin (the NPHS1 gene product) and predicted domain structure. Figure 4A (SEQ ID NO: 2), is the predicted N-terminal signal sequence is 22 residues, the cleavage site being marked with an arrow. A putative transmembrane domain (spanning residues 1,059-1,086) is shown in bold and underlined. The putative extracellular part of the protein contains eight Ig-like modules (boxed), and one fibronectin type III – like module adjacent to the transmembrane domain (boxed with a bold line, residues 941-1025). Cysteine residues are indicated by black dots and the ten putative N-glycosylation sites in the extracellular part of the protein are underlined. Figure 4B shows the predicted domain structure of normal nephrin and the predicted effects of the two mutations (Fin-major and Fin-minor) identified in

this study. The Ig-like modules are depicted by partial circles and the fibronectin type III like-motif by a hexagon. The transmembrane domain is shown as a black rectangle located in a membrane lipid bilayer. The locations of two free cysteine residues are indicated by lines with a black dot at the end. The Fin-major mutation would result in the production of part of the signal peptide and a short nonsense sequence. The Fin-minor mutation would result in a nephrin molecule lacking a part of the cytosolic domain; and

On page 9, please replace the paragraph beginning on line 19, and ending on page 10, line 9, with the following rewritten paragraph:

The NPHS1 gene was analysed by PCR-amplifying and sequencing all exon regions from genomic DNA. The sequences of the primers for exon 2 were 5'GAGAAAGCCAGACAGACGCAG3' (5' UTR) (SEQ ID NO: 3) AND 5'AGCTTCCGCTGGTGGCT3' (intron 2) (SEQ ID NO: 4), and the sequences of the primers for exon 26 were 5'CTCGGGGAGACCCACCC3' (intron 23) (SEQ ID NO: 5) AND 5'CCTGATGCTAACGGCAGGGC3' (intron 26) (SEQ ID NO: 6). PCR reactions were performed in a total volume of 25 ul, containing 20 ng of template DNA, 1x AmpliTaq buffer (Perkin-Elmer), 0.2 mM of each nucleotide, 50 ng of primers and 0.5 U AmpliTaq Gold DNA polymerase. The reactions were carried out for 30 cycles with denaturation at 95° C for 1 min, annealing at 60° C for 1 min, and extension at 72° C for 1 min. In the first cycle denaturation was carried out for 12 min, and extension in the last cycle was for 8 min. PCR products were separated by 1.5% agarose gel, sliced off and purified by the QiaexII system (Qiagen). The purified PCR product was sequenced using specific primers employing dRhodamine dye-terminator chemistry and an ABI377 automated sequencer (Perkin-Elmer).

On page 25, please replace the paragraph beginning on line 14 and ending on line 22, with the following rewritten paragraph:

> With the identification and characterisation of nephrin as a critical component in kidney pathology and proteinuria, and thus implicated in many kidney diseases, it is now possible to screen for small molecule therapeutics using nephrin and the nephrin gene. Screening for such therapeutics can be accomplished by sequential selective screening for activity and molecules which specifically hybridize to nephrin, or which specifically effect the expression of the ~~neprhrin~~ nephrin gene. Selective screening can be performed on pools of small molecule compounds generated by standard combinatorial chemistry, on known ~~moleucles~~ molecules, or in combination with computer modeling of the nephrin protein structure and rational drug design. Such method and techniques are known in the art.